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Date: December 29, 1999

Case No.: 1998-LHC-1103

OWCP No.: 5-29250

In the Matter of :

MILDRED SHEARIN (Widow of
RUDOLPH SHEARIN),
Claimant,
v.

NEWPORT NEWS SHIPBUILDING
AND DRY DOCK COMPANY,
Employer,
and

DIRECTOR, OFFICE OF WORKERS'
COMPENSATION PROGRAMS
Party-in-Interest.

Appearances:

Gary R. West, Esq.
For the Claimant

Benjamin M. Mason, Esq.
For the Employer

BEFORE: Richard K. Malamphy
Administrative Law Judge

DECISION AND ORDER GRANTING SURVIVOR'S BENEFITS
AND DENYING SECTION 8(F) RELIEF

This proceeding arises from a claim filed under the Longshore and Harbor Workers' Compensation Act (the "Act"), as amended, 33 U.S.C. Section901 et. seq.

A formal hearing was held in Newport News, Virginia, on August 25, 1999, at which time all parties were afforded full opportunity to present evidence and argument as provided in the Act and the applicable regulations.

The findings and conclusions that follow are based upon a complete review of the entire record in light of the arguments of the parties, applicable statutory provisions, regulations and pertinent precedent.

STIPULATIONS(1)

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1THE FOLLOWING ABBREVIATIONS WILL BE USED AS CITATIONS TO THE RECORD:

ALJX - ALJ EXHIBITS;
CX - CLAIMANT'S EXHIBITS;
EX - EMPLOYER'S EXHIBITS; AND
TR - TRANSCRIPT OF THE HEARING.

At the hearing, the Claimant and the Employer stipulated as follows:

1. Decedent was employed by the Newport News Shipbuilding and Dry Dock Company as a pipefitter and pipefitter foreman between 1940 and 1980. An employee-employer relationship existed between Decedent and Newport News Shipbuilding and Dry Dock Company for all times pertinent to this claim.

2. Throughout his employment at the Newport News Shipbuilding and Dry Dock Company, Decedent performed work related to ship repair or ship construction aboard ships on the navigable waters of the James River or its adjacent piers and dry docks.

3. The Newport News Shipbuilding and Dry Dock

Company is in the business of constructing and repairing ocean-going vessels.

4. Throughout many years of his employment at the Newport News Shipbuilding and Dry Dock Company, Decedent was exposed to airborne asbestos dust and fibers during and in the course of his employment.

5. On or about October 6, 1996, Decedent died.

6. The Claimant married Decedent on August 8, 1947, and she remained married to, resided with, and was dependent upon Decedent until his death.

7. Decedent retired from active continuing employment more than one year prior to his death. The Claimant and the Employer agree and stipulate that the average weekly wage of Decedent at the time of his death was $400.53 per week, the national average weekly wage at that date.

8. Upon receipt of knowledge that Decedent's death was in part occupationally-related, the Claimant filed a timely claim for benefits under the Longshore and Harbor Workers' Compensation Act, and the Employer filed a timely notice of controversion (ALJX 1).

ISSUES

1. Whether Mr. Shearin had work-related asbestosis that contributed to his death?

2. If so, is the Employer entitled to Section 8(f) relief?

CONTENTIONS

The Claimant contends that Mr. Shearin developed asbestosis due to asbestos exposure with the Employer.

Moreover, the asbestosis was a contributing cause of death.

The Employer agues that death was due to coronary artery disease. However, if it is found that asbestos exposure played a part in Mr. Shearin's death, then Section 8(f) relief should be granted to the Employer as the worker had pre-existing permanent partial disabilities.

The Director states that in the event that it is determined that Mr. Shearin had a permanent disability due to asbestos-related lung disease, the Employer must quantify the level of impairment resulting from this disability alone, and then establish that Mr. Shearin's ultimate level of disability was materially and substantially greater than the disability that would have resulted from the asbestos-related lung disease alone. In this case, the evidence conflicts not as to the extent of Mr. Shearin's lung impairment, but its etiology. If it is determined that Mr. Shearin did have a permanent disability, due to asbestos-related lung disease, the Employer should not be entitled to section 8(f) relief because it has not submitted any evidence that Mr. Shearin's disability was not due to this work condition alone - i.e., the Employer has not produced any evidence that the ultimate disability was substantially greater than that which would have arisen absent the pre-existing disability.

EVALUATION OF THE EVIDENCE

Mr. Shearin died on October 6, 1996. The certificate of death lists the cause of death as cardiac arrest due to an acute myocardial infarction due to coronary artery disease. The certificate was signed by Dr. Horgan (CX 1).

Dr. Horgan was the treating physician during hospitalization from September 5 to October 6, 1996. Clinical data indicated that the worker suffered a myocardial infarction in June, was treated for unstable angina in August, and was now admitted for treatment of increasing shortness of breath and pulmonary endema. Renal failure developed and Mr. Shearin died (CX 3).

On autopsy, the final diagnosis was

> congestive heart failure due to ischemic heart disease with severe calcific coronary atherosclerosis complicated by CMV pneumonia and probable pulmonary asbestosis.
>
> Examination revealed that both lungs appear congested. Cross sections show fine black nodularity of up to 5.0mm each scattered across the lung parenchyma, more prominent in the upper lobes. The bronchi are patent. The pulmonary vessels are negative for thrombi or emboli.
>
> The coronary arteries show multiple foci or moderate to severe calcific atherosclerosis of left and right coronaries with areas of 90% luminal narrowing.
>
> Microscopic evaluation of the lung sections also show numerous ferruginous bodies, anthracotic pigment, pleural and subpleural fibrosis.
>
> The coronary arteries show calcific atherosclerosis with areas of severe luminal narrowing. Subendocardial fibrosis is seen in the left lateral wall myocardium (CX 2).

Dr. Maddox, a pathologist, reviewed the autopsy slides in late 1996. The physician stated that the prosector had reported an enlarged heart and a thickened right ventricle (0.5 cm).

Dr. Maddox reported that:

> Asbestos bodies are numerous, being found embedded in the interstitium and within alveolar macrophages, either individually or in clumps and clusters. These changes correspond to grade 2-3C asbestosis, according to histologic criteria of the C.A.P. Pneumoconiosis Committee. In addition, sections from the heart show old fibrosis in the left ventricle, thickening

> of the right ventricle, and marked atherosclerotic stenosis of the coronary arteries.
>
> Asbestosis caused destruction of part of the pulmonary capillary bed, and this in turn caused increased pulmonary arterial resistance. Increased resistance caused an increase in right ventricular pressure and hypertrophy of the right ventricular myocardium. So cor pulmonale was both a marker of the severity of chronic lung disease and another contributor to the terminal pulmonary edema. Certainly, asbestosis also directly contributed to the loss of pulmonary functional reserve because of decreased transport of gasses across the alveoli.
>
> In summary, with a reasonable degree of medical certainty, this man had clinical and pathologic evidence of relatively high-grade asbestosis that contributed to the development of cor pulmonale and to his death from respiratory failure (CX 4).

Dr. Legier reviewed slides in early 1999. The physician reported a very high burden of asbestos bodies. Cor pulmonale was due to emphysema and asbestosis. Dr. Maddox reported that the moderate degree of asbestos is present, acquired as a consequence of his occupational exposure to asbestos, contributed to his death (CX 5).

In mid 1999, Dr. Roggli, a pathologist, reviewed the materials. The physician stated that:

> Asbestos bodies are noted within the peribronchiolar fibrosis and also within alveolar spaces on hematoxylin and eosin-stained sections. Therefore, this case satisfies histologic criteria for a diagnosis of asbestosis, which we estimate to be grade 1 on a scale of 0-4. (1) A section of the right upper lobe additionally demonstrates a focus of

> cytomegalovirus (CMV) pneumonitis. Other significant autopsy findings include the demonstration of occlusive calcific coronary atherosclerosis and interstitial fibrosis of the ventricular myocardium consistent with a healed myocardial infarct (CX 6).

Dr. Fechner submitted a report in early 1999. The physician stated, in part:

> The lungs contained innumerable asbestos bodies often numbering 10 to 15 in a single high power field. These are located in the alveolar septa as well as in the air spaces. Some asbestos bodies are in histiocytes lying in the air spaces, and other bodies are lying free.
>
> There is fibrosis of the small airways of the type seen in asbestos induced "airways disease." The peribronchiolar fibrosis is accompanied by alveolar (interstitial) fibrosis beyond the periobronchial component, and it is characteristic of asbestosis. The conspicuously high asbestos burden as evidenced by the asbestos fibers is ample to explain the distribution and appearance of the fibrosis.
>
> The role of asbestosis in the fatal outcome of this patient is due to the alveolar fibrosis. The alveolar fibrosis impeded gas exchange and compromised pulmonary function at a time when maximum function was necessary to sustain oxygen in the face of pulmonary edema and CMV pneumonia.
>
> I conclude with a reasonable degree of medical certainty that asbestosis is present. I further conclude with a reasonable degree of medical certainty that the pulmonary function was impaired by asbestosis and was a contributing cause to death of Mr. Shearin (CX 7).

Dr. Stitik conducted a review of X-rays, impressions were:

1. Moderate asbestos-related pleural disease.

2. Minimal diffuse interstitial disease. Whether this is due to asbestosis or other causes is uncertain.

3. Probable area of rounded atelectasis (CX 8).

Following examination in December 1983, Dr. Moore reported the impression of asbestosis (CX 8).

In April 1997, Dr. Horgan sated that Mr. Shearin died from complications of coronary artery disease (EX 2).

Dr. Churg, a pathologist, reviewed records and materials in early 1997. The physician stated that:

> There is definitely no evidence of diffuse interstitial fibrosis in the lung tissue. Focal areas immediately under the pleura demonstrate quite marked and quite shallow fibrosis, but this does not extend into the parenchyma. Centrilobular emphysema is present. In a few of the emphysematous foci and in a few nonemphysematous foci, there is scarring of the small airways in the pattern that we have referred to as "asbestos airways disease" and that others think represents the earliest form of asbestosis. Asbestos bodies are present in the sections. There are also scattered foci of cytomegalovirus pneumonia, undoubtedly a terminal event. The heart shows severe coronary atherosclerosis and evidence of previous myocardial infarction. There is emphysema which is presumably related to cigarette smoking. The small airway lesions, whether one regards them as very early asbestosis or (as I do) another process entirely, are presumably of very little functional consequence in this

> clinical setting and certainly would not mimic the functional consequence of true asbestosis. None of the asbestos-related processes present in this patient's lung would have, in my opinion, contributed in any way to his death (EX 1).

Dr. Ross reviewed records in mid 1981 and again in late 1998. Dr. Ross agreed with Dr. Churg who stated that Shearin did not have asbestosis. Dr. Ross went on to state that:

> It is my firm opinion that neither asbestos nor asbestos exposure played any role in, or made any contribution to, his death as a result of myocardial infarction. His death had no relationship to the findings related to asbestos exposure. Dr. Maddox made an unconvincing effort to explain how asbestosis contributed to his death by causing cor pulmonale. He based this on a slight thickening of the right ventricular wall which he postulated was caused by capillary bed destruction which then led to increased pulmonary resistence and increased pulmonary artery pressure and that those changes helped to cause pulmonary edema. He said that the death was due to respiratory failure but Dr. Horgan said it was due to cardiac failure. Dr. Maddox doesn't seem to have a clear understanding of cardiopulmonary physiology. There was no evidence of the level of capillary destruction required to cause pulmonary hypertension. Cardiac pulmonary edema results from left heart failure. Dr. Maddox's explanation about the development of pulmonary edema and that asbestosis contributed to death in this case is incorrect. There is nothing to indicate that there was any contribution to his death from asbestosis or the other changes related to asbestos exposure. There is nothing to suggest that asbestosis or other results of asbestos exposure

hastened his death. His death was caused by a myocardial infarction as a result of coronary artery disease (EX 3).

DISCUSSION

Exposure to asbestos is acknowledged in this case but the Employer and the Director question the presence of asbestosis. If asbestosis is present, such impairment is conceded to be related to on-the-job asbestos exposure with the shipyard.

The autopsy prosector, Dr. Caplan, suspected the presence of asbestosis. Other pathologists, Drs. Maddox, Legier, Roggli, and Fechner were more adamant in the diagnosis of asbestosis. Dr. Moore shared this impression.

Dr. Churg stated that there was a possibility of early asbestosis. Dr. Ross, who is board certified in Internal Medicine and Pulmonary Disease, and who is a consultant for the employer was clear in stating that asbestosis was not present.

It became apparent during the trial of Nan Parks, Widow of Herman Parks v. Newport News Shipbuilding and Dry Dock Co. (see 32 BRBS 90 (1998)), on appeal to the U.S. Court of Appeals for the Fourth Circuit, that there are some six pathologists, who specialize in the diagnosis of asbestosis. Drs. Churg, Maddox and Roggli were mentioned at that time.

Drs. Maddox and Roggli as well as others have stated that Mr. Shearin had asbestosis. Even Dr. Churg indicated that there might be early signs of such abnormality. I defer to the opinions of the pathologists over those of Drs. Ross and Horgan.

The next question is whether or not asbestosis contributed to Mr. Shearin's death. Drs. Ross and Horgan have clearly stated that there is no relationship.

The autopsy prosector, Dr. Caplan, mentions asbestosis but does not appear to state that this disorder was a contributing cause of death. Dr. Churg clearly

ruled out contribution in this case. Dr. Roggli did not make a definitive statement.

On the other hand, Drs. Maddox, Legier, and Fechner were quite definite in reporting that asbestosis was sufficient in this case to contribute to Mr. Shearin's death. I defer to the above physicians as these pathologists clearly focused on the issue in question and stated that asbestosis played a significant role in the worker's death.

SECTION (8F) RELIEF

Section 8(f) of the Act may be invoked by Employer to limit its liability for compensation payments for permanent disability and death benefits if the following elements are present: (1) Claimant had pre-existing permanent partial disability; (2) the pre-existing disability was manifest to Employer; and (3) the disability or death which exists after the work-related injury is not due solely to that injury, but is a combination of both that injury and the existing permanent partial disability.

In the brief, the Director has stated that:

> The employer asserted in its application for section 8(f) relief that Mr. Shearin had the pre-existing conditions of hypertension/coronary artery disease and COPD, based on Dr. Reid's report. Employer's Exhibit (4(c). In light of Dr. Horgan's reports, and since coronary artery disease is listed as a cause of death on Mr. Shearin's death certificate, the Director agrees with the employer's assertion of these pre-existing conditions.
>
> In light of the fact that there was a diagnosis of hypertension/coronary artery disease and COPD from shipyard clinical records and Dr. Horgan, the Director agrees with the employer's assertion that these pre-existing conditions were manifest prior to the diagnosis of asbestosis.

> Accordingly, the employer has established the manifestation element of its claim for section 8(f) relief. (See Newport News Shipbuilding and Dry Dock Co. v. Harris, 934 F.2d 548 (4th Cir. 1991)).

CONTRIBUTION

Pursuant to 20 C.F.R. Section702.321(a)(1), if an employer is seeking Section 8(f) relief for survivor's benefits, the medical report must establish that the death was not due solely to the second injury. See 20 C.F.R. Section702.3219a)(1). Newport News Shipbuilding must show that the pre-existing permanent partial disability materially and substantially contributed to death. Newport News Shipbuilding can satisfy this contribution element by proving that the employment-related injury aggravated the pre-existing condition. See Vlasic v American President Lines, 20 BRBS 188, 192 (1987). Section 8(f) relief, however, is not applicable when the claimant's disability results from the progression of, or is a direct and natural consequence of, the pre-existing disability. See Director, OWCP v. Cooper Associates, Inc., 607 F.2d 1385, 10 BRBS 1058 (D.C. Cir. 1979).

In March 1997, Dr. Reid, a physician in the shipyard clinic, reviewed records and prepared a report. The physician stated that:

1. Mr. Shearin had pre-existing hypertension/arteriosclerotic heart disease/coronary artery disease and chronic obstructive pulmonary disease long before he was diagnosed to have asbestosis after his death on October 6, 1996.

2. Mr. Shearin had long-standing permanent and significant hypertension and related coronary artery disease. As long ago as 1973, Mr. Shearin was diagnosed to have hypertension (EX 1 and 2). By June 1996, his hypertensive

cardiovascular disease had developed into severe coronary disease, with a resultant myocardial infarction (heart attack), which by definition means part of the heart muscle died (EX 3 and 4).

3. As early as 1981, Mr. Shearin was noted to have "chronic obstructive pulmonary disease" ("COPD"), which is a permanent and serious lung disease which causes significant obstructive lung impairment (EX 2).

4. Mr. Shearin's pre-existing conditions of hypertension/coronary artery disease and COPD were permanent and serious. A cautious employer would not hire a worker for heavy manual labor, such as at the Shipyard, if he had either condition.

5. Mr. Shearin's pre-existing conditions of hypertension/coronary artery disease was manifest based on the records of Dr. Ross and Dr. Horgan.

6. Mr. Shearin's death was caused by his heart disease (EX 5). Even if Mr. Shearin had some pathological asbestosis, it did not cause or hasten, even in part, Mr. Shearin's death. If Mr. Shearin merely had asbestosis, he would clearly be alive today. Even if Mr. Shearin had asbestosis, it was not of clinical significance. Mr. Shearin died of his heart disease - "cardiac arrest."

[The exhibits cited by Dr. Reid do not correlate to those of record in this case]

The Director argues that none of the medical evidence submitted by the Employer in conjunction with its

application for section 8(f) relief quantifies the level of impairment resulting from the Claimant's work-related injury alone. Thus, the Employer has not met its burden of offering evidence from a doctor, such as a treating physician, who could testify to the extent and seriousness of the work-related injury and the degree of disability it would have caused alone. Director, OWCP v. Newport News Shipbuilding and Dry Dock Co. (Carmines), 138 F.3d at 143-44 (4th Cir. 1998).

The Employer argues that the medical reports quantify the amount of Shearin's disability from his pre-existing conditions alone. Finally, each satisfies the fundamental requirement of Section 8(f) relief that the ultimate disability not follow from an industrial injury alone. Indeed , the Employer has not only proved contribution, but it has also shown that the majority of Mr. Shearin's impairment resulted from his pre-existing COPD, hypertension and heart disease.

The undersigned finds that the Director is correct and the shipyard is incorrect in assessing the burden of contribution. Carmines, in citing Director, OWCP v. Newport News Shipbuilding and Dry Dock Co. (Harcum), 8 F.3d 175 (4th Cir. 1993), states that there must be a determination of the degree of disability resulting from the work related injury alone.

The Employer focuses on the extent of pre-existing disability. In fact, Dr. Ross, the shipyard physician, stated that asbestosis was not of clinical significance.

I conclude that the Employer has not met the criteria for contribution pursuant to Carmines and Harcum.

ORDER

It is hereby ORDERED that:

1. The Employer is to pay death benefits to the surviving spouse from October 6, 1996, to the present and continuing at the compensation rate of $400.53 x 1/2 per week.

2. The Employer is to pay to Mrs. Shearin $3,000.00 for funeral expenses pursuant to Section 9(a) of the Act.

3. Employer is to pay to the Claimant interest at the rate specified in 28 U.S.C. Section 1961 in effect when this Decision and Order is filed with the Office of the District Director. Interest shall be paid on all accrued benefits computed from the date on which each payment was originally due to be paid. See Grant v. Portland Stevedoring Co., 16 BRBS 267 (1984).

4. All computations are subject to verification by the District Director.

5. The application for Section 8(f) relief is DENIED.

6. Claimant's attorney, within twenty (20) days of receipt of the order, shall submit a fully supported fee application, a copy of which shall be sent to opposing counsel, who shall then have ten (10) days to respond thereto.

_____RICHARD K.
MALAMPHY

Administrative
Law Judge

RKM/dlh
Newport News, Virginia